
Exercise-induced acute compartment syndrome in a young man, occurring after a short race

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We describe a case of exercise-induced acute compartment syndrome (ACS) in a 23-year-old man who presented to his primary care physician 48 hours after he attempted to run a 5K race. He noticed searing pain in his left leg after the first half mile but had no other symptoms. He was referred to the emergency department and diagnosed with ACS, and a fasciotomy was done. A presentation of limb pain that is out of proportion to a known or suspected injury should prompt consideration of ACS. Early recognition and surgical management are essential to achieving the best possible outcome.

Acute compartment syndrome (ACS) is an emergency requiring immediate surgical decompression (1). Because it can progress rapidly, urgent diagnosis and treatment are necessary to prevent subsequent tissue ischemia and necrosis. The syndrome is typically a consequence of trauma (2). Chronic compartment syndrome is a common problem among athletes, while an acute presentation after exercise is rare (3–7). Our case demonstrates delayed presentation of exercise-induced ACS.

CASE PRESENTATION

A 23-year-old man with no significant medical history presented to his primary care physician with severe pain in the left lower leg. Two days earlier, he attempted to run a 5K race but had to stop after the first half mile. He complained of searing pain associated with numbness in his left lower extremity. He described some pain in his right leg as well, but of a less severe nature. He stopped running with the onset of pain and attempted to resume after stretching but was unable to do so due to his pain. Rest, warm and cold compresses, acetaminophen, and nonsteroidal antiinflammatory drugs did not alleviate the pain. On examination, he had significant swelling and tenderness in the anterior and lateral aspects of his left leg. Distal pulses were palpable in both legs. He was sent to the emergency department for further evaluation.

Radiographs taken in the emergency department showed no bony injury or other abnormality. Doppler venous ultrasound revealed no evidence of deep vein thrombosis. His serum creatine kinase was 15,988 U/L (normal range, 30–233); aspartate aminotransferase, 348 U/L (normal range, 13–59);

alanine aminotransferase, 164 U/L (normal range, 7–53); and creatinine, 1.19 mg/dL (normal range, 0.7–1.3). Compartment pressures in the leg were obtained using a commercially available intracompartmental pressure monitor system and found to be 50 in the anterior and lateral compartment with a delta pressure of <30. A left anterior and lateral fasciotomy was performed within 6 hours of initial presentation. No muscle rupture was found intraoperatively. Aggressive intravenous fluid therapy was initiated to treat his rhabdomyolysis. Postoperatively, the patient's symptoms improved rapidly and his serum creatine kinase quickly improved. Two days later, secondary closure of the skin overlying the fascial releases was performed. The patient was discharged home after a 3-day stay.

DISCUSSION

ACS is a limb-threatening and occasionally life-threatening condition characterized by increased interstitial pressure within an osteofascial compartment, exceeding a critical point and resulting in reduced tissue perfusion and myoneural ischemia (8). ACS has been reported after heavy exercise, weight lifting, military training, and marathons (8–10). Our patient had leg pain after a very short run. The differential diagnosis included compartment syndrome, muscle group rupture, stress fracture, and ligament injury. We used a noninvasive test to determine compartment pressure. A recent metaanalysis suggested use of intracompartmental pressure measurement to confirm the diagnosis in patients in whom ACS is suspected (11). Magnetic resonance imaging of the leg would have helped with the preliminary differentials, but with an elevated delta pressure of the lower extremity, ACS was considered and emergency fasciotomy was done.

ACS should be especially suspected in the presence of one or more of the following symptoms: pain out of proportion to a known or suspected injury (with or without pain on passive stretch), sensory changes, and a loss of motor power (12).

In our patient, ACS is likely to have occurred secondary to exercise-induced rhabdomyolysis resulting in tissue edema

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with subsequent muscle injury and tissue damage. Some cases of exercise-induced compartment syndrome may evolve over a prolonged period of time, with slow but progressive muscle tissue injury. The fascia that delineates the compartments prevents expansion, causing a rise in the intracompartmental pressures (13). This can cause further elevations in creatine kinase, which can lead to severe acute kidney injury, electrolyte abnormalities, and permanent muscle damage. In our patient, the creatine kinase level was elevated to >10,000 without evidence of acute kidney injury or myoglobin in urine.

Rhabdomyolysis must be considered in patients presenting with ACS so that early compartment release surgery and vigorous fluid resuscitation can be initiated. Elevated serum transaminases were also noted at presentation, a finding that improved in parallel to the creatine kinase levels. The patient had no high-risk behavior, and serology for hepatitis viruses was negative. Skeletal muscle injury is believed to be the most likely source of the elevated transaminases.

ACS is diagnosed on the basis of clinical findings. In high-risk patients, surgical consultation, possibly including the measurement of compartment pressures, should not be delayed in order to obtain laboratory test results. A delta pressure (compartment pressure subtracted from diastolic blood pressure) of <30 mm Hg indicates a need for fasciotomy (14). Serial or continuous measurements are essential when patient risk or clinical suspicion is high.

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